Draft Updated
Human Health Risk Assessment
for Dick's Creek and Tributaries

AK Steel
Middletown, Ohio

Submitted to:

U.S. Environmental Protection Agency
Region 5
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Acronym List

AT	Averaging time
BW	Body weight
C	Chemical concentration
CR	Contact rate
CTE	Central tendency exposure
ED	Exposure duration
EF	Exposure frequency
EPC	Exposure point concentration
HHRA	Human Health Risk Assessment
HUS	Human Use Survey
I	Intake
NOAEL	No observed adverse effect level
PAHs	Polycyclic aromatic hydrocarbons
PCB	Polychlorinated biphenyls
PCDF	Polychlorinated dibenzofurans
RfD	Reference dose
RME	Reasonable maximum exposure
CSF	Cancer slope factor
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TEQ	Toxicity equivalence factors
UCL	Upper confidence limit
WSU	Wright State University

1.0 Introduction

This draft document updates the *Draft Human Health Risk Assessment Dick's Creek and Tributaries AK Steel* (HHRA), dated November 17, 2000 and prepared by Tetra Tech EM Inc. (TT) for the AK Steel, Middletown Works facility, Middletown, Ohio. As part of this update, USEPA Region 5 requested a detailed review of the following documents:

- The TT HHRA. In order to identify which parts of the risk assessment require revision based on updated and more current risk assessment methodology for poly-chlorinated biphenyls (PCBs), as well as recently collected environmental data.

- The *Human Health Risk Assessment, Dick's Creek Middletown, OH,* dated June 4, 2001 and prepared by Arcadis G&M (AGM) for Frost Brown Todd LLC and AK Steel. To evaluate the risk assessment methodology and the datasets that were used to quantify human health risks, as well as the veracity of the risk estimates.

- The comprehensive list of datasets for all environmental media collected to date. To evaluate sampling and analysis protocols, overall data quality, representativeness for quantifying exposure, and chemical dose. Of primary interest is the dataset generated by Wright State University (WSU), which indicated that levels of the highly toxic dioxin-like PCBs were significantly elevated in Dick's Creek.

Based on the detailed review of the above mentioned documents and information, it was concluded that:

- The risk assessment methodology must be updated for both TT and AGM risk assessments to follow the USEPA guidance *PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures. Office of Research and Development*, (USEPA 1996) that has been specifically developed for uncontrolled releases of PCBs. This USEPA guidance contains many of the same elements developed by the National Academy of Sciences, National Research Council (NRC), which are presented in *A Risk-Management Strategy for PCB-Contaminated Sediments* (NRC 2001). The methodology used in both the TT and AGM risk assessments is very general, and is applicable only for estimating risks for single chemicals. It is not applicable or appropriate for evaluating complex mixtures of PCBs, which comprise individual PCB congeners of varying toxicity that undergo unique partitioning in the environment due to fate and transport mechanisms, and weathering.

- Environmental data for the most toxic constituents—namely, the PCB dioxin-like congeners—is lacking. Consequently, human health risks cannot currently be precisely estimated. At best, the current risks (including those in this updated report) should be considered only a portion of the total risks posed by the uncontrolled release of PCBs. USEPA 1996 PCB risk assessment guidance outlines a tiered approach that needs to be followed in which risks are estimated based on the total PCB concentration detected in all environmental media. The dioxin-like PCB risks are then added to the risks estimated for total PCBs. Because Aroclor 1248 has been detected, it is likely that many dioxin-like congeners are also present but have been ignored in sampling and, consequently, in the risk assessment. For example, Shwartz *et al.* (1993) has shown that a small group of dioxin-like PCBs is concentrated in Aroclor 1248, which has been routinely detected in Dick's Creek sediment samples. This is important because those dioxin-like congeners are more than a thousand-fold toxicologically potent than non-dioxin-like PCBs. USEPA guidance states that Aroclor data should not be used to quantify PCB-related risks because, after weathering and partitioning occur, PCBs can go undetected even though they may be present in high concentrations. For this reason, the non-detect values reported in the Aroclor datasets from Dick's Creek and tributaries are suspect. The extent of human health risks will remain questionable (although likely underestimated) until appropriate PCB congener data becomes available.

- While dioxin-like PCB data do not currently exist in USEPA or AK Steel databases, WSU has sampled a variety of environmental media and analyzed for dioxin-like PCB congeners. Unfortunately, the quality of this dataset could not be verified in time to prepare this updated HHRA. When the quality of the data is validated, this current updated HHRA should be further updated to quantify those risks.

- While risks calculated for non-PCBs—polycyclic aromatic hydrocarbons (PAHs) and metals (arsenic)—were shown to be high, they are likely to be unrelated to site releases. Although the risks were verified to be correctly calculated, they represent either naturally occurring or anthropogenic and ambient regional conditions. Consequently, no further risk assessment analysis was deemed necessary to update this report for non-PCB chemicals.

- AK Steel used a brief survey of exposures (which is essentially a snapshot in time) to develop both current and future exposure conditions upon which their risk is estimated. AK Steel cannot legally enforce exposure conditions off their property and cannot guarantee that the survey conditions will not change in the future. Therefore, it is prudent in order to protect public health to assume exposures in Dick's Creek will resemble exposures throughout the United States for which there is an extensive and peer-reviewed statistical data base for exposure conditions.

- The more recent sampling data, collected by AK Steel, should be combined with the previous datasets used in the TT HHRA. This will provide additional data to both increase the number of samples, which will increase the statistical confidence and representativeness for estimating exposure conditions, and will provide additional temporal information regarding the change in PCB concentrations since the last samples were collected.

- The exposure assumptions and Aroclor data used in the TT HHRA to estimate human health risks for all chemicals regarding exposure to contaminated surface water and fish were individually verified and are reasonable, based on USEPA guidance. However, the dioxin-like PCB risks estimated by TT, which are based on WSU data, could not be verified due to lack of information on the quality of the WSU dataset. No new pertinent surface water and fish data were available to update exposure to those pathways. However, numerous AK Steel samples are now available for sediment contamination to combine with the previous dataset used in the TT HHRA.

1.1 Report Purpose and Organization

The goal of this draft updated HHRA is to quantify, to the extent possible, potential risks to human health associated with exposures in Dick's Creek and its tributaries attributable to uncontrolled releases of PCBs from the AK Steel facility. The risks presented in this HHRA are based on current and hypothetical future exposure conditions in the absence of remediation efforts or institutional controls. It is organized into the following sections:

- Site Characterization
- Data Evaluation and Estimating the Exposure Point Concentrations
- Exposure Assessment
- Toxicity Assessment
- Risk Characterization, and
- Uncertainty Assessment.

The overall risk assessment methodology for PCB-contaminated sites is different from the approach used at sites where risks for individual chemicals are estimated and simply summed. USEPA PCB risk assessment guidance (USEPA 1996) requires a tiered approach in which risks are estimated based on total PCB concentration detected in each sample. PCBs are manmade, highly complex mixtures of 209 individual congeners. Each of these congeners has a distinct chemical property and inherent toxicity that is due to the number and placement of chlorine atoms on a biphenyl ring. The term Aroclor simply refers to the trademark commercial mixtures with varying amounts of these different congeners in the *original* mixture. All PCB congeners released into the environment will partition into different environmental media (i.e., water, soil, air, animals, etc.) based on the chemical properties of each *congener*. Consequently, USEPA guidance specifically requires the use of three different toxicity values, which are based on individual *environmental media* and *exposure routes* to estimate risks, rather than on specific Aroclors (i.e., Aroclor 1248, 1260, etc.)

2.0 Site Characterization

The site is well characterized with regard to the AK Steel operational areas, transport mechanisms, Dick's Creek, and its tributaries in the TT and AGM HHRAs. Those documents should be reviewed for background information on the site in general.

The first step in developing a framework for the risk assessment is defining the area over which exposure will routinely occur for the same individual. USEPA (1989) guidance states:

> *"The area over which the activity is expected to occur should be considered when averaging the monitoring data for a hot spot. For example, averaging soil data over an area the size of a residential backyard (e.g., an eighth of an acre) may be most appropriate for evaluating residential soil pathways."*

Likewise, for a recreational exposure, it is reasonable to assume, due to human nature, that individuals who frequent Dick's Creek and its tributaries will find a favorite spot and habitually return to the same location. It is unreasonable to assume that an individual will be exposed to the entire length of Dick's Creek and its tributaries during the day exposures occur. Consequently, to estimate the average daily dose of PCBs, data must be aggregated over the area where exposure is expected to occur for an individual. The representative PCB concentration resulting from aggregating data over the exposure area or unit of exposure is termed the exposure point concentration (EPC). In this updated HHRA, both TT and AGM HHRAs were reviewed to determine the most appropriate approach for defining exposure areas along the river in order to estimate the EPC. While the TT HHRA defines several discreet and reasonable length sections of Dick's Creek and its tributaries as individual exposure units, the AGM report assumes an individual will be exposed to the entire length of the river and its tributaries during the day. While it is plausible a jogger or hiker could be exposed to the entire length, these receptors are not the type of individuals for which this updated HHRA has been conducted. The receptors currently exposed and who are expected to be exposed in the future are similar to those visually identified at Dick's Creek. Children, adolescents, and adults have been directly observed recreating at the river. Furthermore, evidence of their activities (e.g., tire swing along the Dick's Creek bank, bag of caught and discarded fish) were observed on a recent (June 5, 2002) site visit, indicating that recreators spend considerable time in discreet short stretches of Dick's Creek and its tributaries. Therefore, a similar framework of exposure units to those developed in the TT HHRA was developed for this update. All datasets, including those presented in the TT and AGM HHRAs, and *Data Summary Report: Sediment and Surface Water* dated April 26, 2001 prepared by AGM, were culled to identify data that are considered representative and could be aggregated to calculate an EPC, based on their reported sampling locations within one of five sections of the river, which are as follows:

- Dick's Creek 2 (DC2): Approximately 200 feet upstream of outfall 2 to Yankee Road.
- Dick's Creek 3 (DC3): Yankee Road to Hamilton Middletown Road.

- Dick's Creek 4 (DC4): Hamilton Middletown Road to approximately 100 feet downstream of Main Street.
- Dick's Creek 5 (DC5): 100 feet downstream of Middletown Road to approximately 4,000 feet downstream of Main Street.
- Landfill Tributary: Tributaries including Monroe Ditch, approximately 3,000 feet from Dick's Creek.

All data identified within these exposure areas were aggregated to estimate the EPC for each section of the river to estimate the dose of PCBs that would result from exposure.

3.0 Data Evaluation and Estimating the Exposure Point Concentrations

Numerous environmental samples have been collected in Dick's Creek surface water, sediments, soil, and fish over the years. With few exceptions, all samples identified as pertinent have been analyzed only for Aroclors despite USEPA guidance (USEPA 1996) strongly urging that Aroclor data *not* be used to quantify PCB-related risks. Consequently, the risks in this report were limited to risks defined by Aroclor data.

One flaw in using Aroclor data is that risks can be overestimated if Aroclor concentrations are simply summed, due to double counting individual congeners that may be present in more than one sample. This did not appear to be a significant problem in this update because relatively few samples reported detected values for more than two Aroclors. However, one important modification made in the data assessment should be noted. In this updated HHRA, nondetects were treated explicitly as nondetects and were *not* assigned a proxy value of one-half the detection limit as is conventional in non-PCB HHRAs. Rather, nondetected values were assigned a value of zero. This modification was based on professional judgment, primarily to avoid double counting congeners. If one-half the detection limit is assigned to each nondetect and substituted for each Aroclor of the entire suite of Aroclors for all nondetect Aroclors in each sample, congeners that are common to two or more Aroclors would have been double counted. However, it should be noted that risks can also be underestimated when the PCB mixtures in environmental samples have undergone weathering, which can transform the original Aroclor mixture released into the environment and make it appear as though PCBs are not present and are denoted as nondetects in samples when, in fact, some highly toxic PCB congeners may be present at high concentrations. This is because when samples are analyzed for Aroclors, the analysis and identification of Aroclors is based on the presence of a characteristic, but limited, subset of PCB congeners that are considered a fingerprint of the Aroclor mixture. Relying on a subset to represent the whole mixture and using it as an approximation of the chromatogram reference profile of the original technical mixture can lead to a subjective interpretation by the analytical chemist. When weathering occurs, some individual congeners are degraded or have partitioned into other environmental media. This concept is explicitly stated in USEPA PCB risk assessment guidance (USEPA 1996):

"Although environmental mixtures are often characterized in terms of Aroclors, this can be both imprecise and inappropriate. Qualitative and quantitative errors can arise from judgments in interpreting gas chromatography/mass spectrometry (GC/MS), which reveals a spectrum of peaks that are compared with characteristic patterns for different Aroclors. For environmentally altered mixtures, an absence of these characteristic patterns can suggest the absence of Aroclors, even though some congeners are present in high concentrations."

As is also noted in the USEPA IRIS file (USEPA 2002) for PCBs, congener analysis is important for the assessment of human health risks posed by a site:

"Although PCB exposures are often characterized in terms of Aroclors, this can be both imprecise and inappropriate. Total PCBs or congener or isomer analyses are recommended."

Accordingly, this updated HHRA (as well as the TT and AGM HHRAs) should be viewed as incomplete until it can be verified, either by additional sampling and analysis or a re-review of the original chromatograms, that the samples with reported nondetect values do not contain unreported PCBs. It should also be noted that weathering preferentially degrades PCB congeners with the lowest toxicity, causing the weight percentage in the samples of more toxic congeners, particularly those that produce dioxin-like effects, to increase on a weight percentage basis, which USEPA (1996) discusses in its PCB guidance:

"Unfortunately, the environmental weathering of Aroclors modulates mixture toxicity (Quensen et al. 1998). As such, carcinogenic risk-assessment guidelines recommend the calculation of congener-specific or total PCB data when available (EPA 1994c). Congener-specific analyses utilize the direct quantification of each unique PCB congener. The result is a precise description of PCB profiles, which can highlight physiological, spatial, and temporal changes that might not be apparent in Aroclor values....Individual congener data provides the most flexibility for supporting environmental management decisions, because the congeners provide the raw data that can be analyzed numerically or statistically by the environmental manager, case by case, as needed.... Congener-specific analysis is recommended for risk assessment because of the differences in the toxic potentials of individual congeners in technical mixtures."

Both TT and the AGM indicate that only data collected after 1998 was used to estimate risks, because these data reflect current, post-remediation conditions. This is contrary to the purpose of a risk assessment, which is to determine the lifetime risks and health effects associated with AK Steel's uncontrolled release, not to arbitrarily and narrowly evaluate risks that exist post-remediation. While it is acceptable to use data that represent current conditions in the risk assessment, it is unacceptable to simply ignore pre-1998 PCB exposures and pretend no exposures occurred prior to 1998 unless AK Steel can clearly demonstrate no exposures actually occurred. Assuming pre-1998 exposures did occur, those PCBs would still, for the most part, be sequestered in the body fat of recreators, where they will remain in the fat deposits for many decades and pose the risk of causing cancer. Pre-1998 data should be used to estimate the lifetime cancer risks since the inception of the uncontrolled PCB release. However, risks should not be overestimated by only using data collected prior to 1998. Rather, the estimated daily intake should be a weighted average over the period of time a representative receptor will reasonably be exposed. For this updated risk assessment, pre-1998 data were included in estimates of exposure. It is reasonable to assume that, since receptors could only be exposed to those pre-1998 concentrations for a fraction of the 30 year exposure duration, the reported concentrations for 1998 were divided by 5 to represent a presumed weighted average for the years exposed to that concentration. Although this approach was based on professional judgment, it satisfies the requirement that as many samples as possible be used to represent both spatial and temporal aspects of exposures. To simply assert exposures did not occur prior to 1998 is not only unacceptable for risk assessment purposes, but it defies common sense.

Data that were culled from all datasets were aggregated according to the corresponding exposure unit (described above) along Dick's Creek and tributaries. All of the EPCs estimated for all environmental media in the TT report were recalculated. Based on this extensive reevaluation, it was concluded that, with the exception of sediment data, all environmental media EPCs were reasonable and represented exposure conditions at the site. Accordingly, those EPC values for surface water and fish that were used in the TT report remained unchanged in this updated HHRA. However, many more pre-1998 data and data generated after the TT HHRA was completed were used to update the HHRA to represent the temporal changes in exposure conditions. Only those samples collected at depths of 0-6 inches were used to calculate EPC values. The upper 95% confidence limit on the mean concentration (95UCL) was calculated based on the default assumption that the underlying distribution of the datasets were lognormal. However, in many instances, the 95UCL exceeded the maximum detected concentration. Therefore, the lower value of either the maximum detected value and the 95UCL was used to estimate risks.

Tables 1 through 5 present the data culled from all available datasets that were used to estimate the 95UCL EPC for each exposure unit section along Dick's Creek and tributaries. Table 6 displays the maximum detected concentration and 95 UCL for each media, and identifies which values were used as the EPC.

TABLE 1

SAMPLES AGGREGATED TO ESTIMATE EXPOSURE POINT CONCENTRATION

SEDIMENT, DC 2

Sample Number	Type of Aroclor	Concentration
02SD01-06	1242	2.80
DCSD15-06	1242	0.12
DCSD16-06	1248	0.79
DCSD17-06	1242	1.10
MDSD01-06	1248	14.00
MDSD02-06	1248	0.11
MDSD03-06	1248	0.12
MDSD04-06	1248	1.20
"	1254	0.35
MDSD05-06	1248	0.26
"	1254	0.07
OEPA-32296	1254	0.07
"	1260	25.40
OEPA-08843	1242	1.40
"	1248	1.40
"	1260	0.30
OEPA-08870	1248	25.80
"	1260	1.70
AK Steel 9601221	1242	0.70
OEPA-10817	1248	0.79
AK Steel 528041	1242	1.40
"	1248	0.25
AK Steel 528041	1242	1.86
"	1248	0.25

TABLE 2

SAMPLES AGGREGATED TO ESTIMATE EXPOSURE POINT CONCENTRATION

SEDIMENT, DC 3

Sample Number	Type of Aroclor	Concentration
DCSD07-06	1242	1.30
"	1254	0.67
"	1260	0.06
DCSD08-06	1242	1.10
"	1254	0.61
"	1260	0.11
DCSD09-06	1242	0.25
DCSD09A-06	1242	0.86
"	1254	0.41
"	1260	0.12
DCSD10-06	1242	0.44
DCSD11-06	1242	1.60
"	1254	0.65
DCSD12-06	1242	0.25
"	1242	0.31
DCSD13-06	1242	0.13
DCSD14-06	1242	0.06

TABLE 3

SAMPLES AGGREGATED TO ESTIMATE EXPOSURE POINT CONCENTRATION

SEDIMENT, DC 4

Sample Number	Type of Aroclor	Concentration
DCSD05-06	1242	0.27
"	1254	0.24
"	1260	0.05
DCSD06-06	1242	1.20
"	1254	0.90
"	1260	0.18
AK-528042	1242	2.01

TABLE 4
SAMPLES AGGREGATED TO ESTIMATE EXPOSURE POINT CONCENTRATION

SEDIMENT, DC 5

Sample Number	Type of Aroclor	Concentration
DCSD01A-06	1242	0.23
DCSD01B-06	1254	0.17
DCSD02-06	1242	0.21
"	1254	0.25
"	1260	0.04
DCSD03-06	1242	0.22
DCSD04-06	1248	0.24

TABLE 5

SAMPLES AGGREGATED TO ESTIMATE EXPOSURE POINT CONCENTRATION

LANDFILL TRIBUTARIES-MONROE DITCH SEDIMENT

Sample Number	Type of Aroclor	Concentration
EPA 99WM03S04	1242	16.80
EPA 99WM03S06	1248	16.00
OEPA 21111-4	1242	7.57
"	1260	3.28
OEPA 21111-3	1242	11.70
"	1260	2.27
OEPA 08842	1242	22.70
	1248	22.70
	1260	7.20
OEPA 08845	1242	14.40
	1248	14.40
	1260	4.10
AK Steel 9600921	1242	0.36
AK Steel 9600922	1242	0.31
AK Steel 9600979	1242	5.30
AK Steel 479120	1248	1.93
AK Steel 479121	1248	0.95
OEPA 10818	1248	32.30
OEPA 17258	1248	14.20
AK Steel 479141	1248	21.00
OEPA 7924	1248	0.87
"	1260	0.29
OEPA 7990	1248	2.70
AK Steel 479105	1248	2.79
AK Steel 479106	1248	2.58
AK Steel 479113	1248	1.29
AK Steel 479117	1248	5.13
AK Steel 479119	1248	4.36

TABLE 6

MEDIA SPECIFIC EXPOSURE POINT CONCENTRATIONS FOR EACH SEGMENT OF DICK'S CREEK

Site	Analyte	Maximum Detected Concentration	95 UCL
DC 1		No values	
DC 2 Surface Water		No values	
DC 2 Sediment	PCB 1	No values	
	PCB 2	2.80e+00	6.29e+00
	PCB 3	2.58e+01	4.70e+01
	Total PCBs	2.75e+01	3.07e+01
DC 2 Fish	PCB 3 (=total)	2.65e+04	2.29e+04
DC 3 Surface Water		No values	
DC 3 Sediment	PCB 1	No values	
	PCB 2	1.30e+00	1.85e+00
	PCB 3	1.60e+00	3.11e+00
	Total PCBs	2.25e+00	1.17e+01
DC 3 Fish		No values	
DC 4 Surface Water		No values	
DC 4 Sediment	PCB 1	No values	
	PCB 2	2.01e+00	2.33e+04
	PCB 3	9.00e-01	9.42e+02
	Total PCBs	2.28e+00	5.73e+02
DC 4 Fish	PCB 3 (=total)	2.00e+03	2.05e+03
DC 5 Surface Water		No values	
DC 5 Sediment	PCB 1	No values	
	PCB 2	2.30e-01	2.40e-01
	PCB 3	2.50e-01	1.41e+01
	Total PCBs	5.00e-01	4.70e-01
DC 5 Fish		No values	
LT Surface Water		No values	
LT Sediment	PCB 1	3.63e+00	1.43e+00
	PCB 2	2.27e+01	4.47e+01
	PCB 3	3.23e+01	7.08e+00
	Total PCBs	2.19e+01	2.16e+01
LT Fish		No values	

Notes: The smaller of the maximum detected concentration and the 95 UCL was used as the exposure point concentration (EPC). The value used is highlighted by double lines.
For fish, there were values only for PCB 3; therefore, these values are equivalent to total PCBs.

4.0 Exposure Assessment

The exposure assessment included an evaluation of potential human receptors that currently contact or are expected to contact PCBs in Dick's Creek and its tributaries, as well as the possible routes, magnitudes, frequencies, and durations of exposure. The primary goal of an exposure assessment is to quantify the average daily dose of PCBs for each potential receptor.

The following are steps taken to develop the exposure assessment:

- Characterize the exposure setting and identify potential current and potential future human receptors;
- Identify complete exposure pathways and routes of exposure for each potential receptor;
- Estimate exposure point concentrations based on using each sample location as an exposure point;
- Quantify chemical intake for individual exposure pathways for each potential receptor; and
- Combine chemical intakes across exposure pathways for each potential receptor.

This paradigm for evaluating exposure follows USEPA guidance. The TT HHRA was based on a similar approach. After careful review of each exposure parameter in the TT HHRA, it was concluded that all were consistent with USEPA exposure databases or based on reasonable professional judgment. Accordingly, the same exposure values were used in this report.

The following equation and generalized exposure parameters are used to estimate exposure conditions at Dick's Creek:

$$\text{Intake (I)} = C*CR*EF*ED*FI*(1/BW)*(1/AT)$$

I	Intake (milligram per kilogram body weight - day, [mg/kg-day])
C	Chemical concentration in contaminated medium (milligram per kilogram [mg/kg] or milligram per liter [mg/L])
CR	Contact rate or ingestion rate (milligrams per day or liters per day)
EF	Exposure frequency; how often exposure occurs (days per year)
ED	Exposure duration; how long exposure occurs (years)
FI	Fraction ingested (unitless)
BW	Body weight (kilogram [kg])
AT	Averaging time; period over which exposure is averaged (days)

Chemical intakes were estimated for each age group—namely, child (aged 0 to 6), adolescent (aged 7 to 12), and adult recreators—for each exposure pathway. The complete exposure pathways for contact with uncontrolled releases of PCB in Dick's Creek and its tributaries are as follows:

- Sediment ingestion
- Dermal absorption of PCB-contaminated sediments
- Ingestion of surface water
- Dermal contact of PCB-contaminated surface water, and
- Ingestion of PCB-contaminated fish.

The specific exposure factors for each of these pathways for each receptor are presented in Appendix A.

According to USEPA guidance (1989), exposure parameters used to estimate contaminant intakes for a given pathway should be selected so that the combination of all intake variables results in an estimate of the reasonable maximum exposure (RME) for that pathway. Standard default assumptions were used to estimate chemical intakes for each route of exposure (USEPA 1989, 1991a, and 1991b).

It should be noted that a detailed review of the AGM HHRA indicated the AK Steel report is based on a relatively brief, unconfirmed Human Use Survey (HUS) of exposure in areas of Dick's Creek. A major flaw in the approach is an over-reliance on the results to develop site-specific exposure parameters, which were used to estimate the chemical dose, or average daily intake. At best, the HUS can be considered a snapshot of *current* human activity and may or may not accurately reflect current conditions, as well as future exposure conditions. Furthermore, the results should only be used to *qualitatively* evaluate current exposure conditions or to estimate the lower end of the range of potential risks. It cannot be used to evaluate future exposure conditions in estimating future risks since AK Steel has no means to legally enforce that current exposure conditions are maintained in perpetuity or at least until PCB levels attenuate to levels that will not pose unacceptable risks. Furthermore, the HUS was conducted while an advisory was in place cautioning "UNSAFE WATER, DO NOT SWIM, BATHE, DRINK, OR FISH," which could temporarily attenuate exposures (indicated in some survey results), but human nature may propel nearby residents to ignore such warnings. Indeed, on the June 5, 2002 site visit to the Dick's Creek areas, evidence of numerous exposures were observed that were in clear violation of posted warnings. According to USEPA (1991c), risk assessments should not be conducted under the assumption institutional controls will be heeded:

> *"The cumulative site baseline risk should include all media that the reasonable maximum exposure scenario indicates are appropriate to combine and should not assume that institutional controls or fences will account for risk reduction."*

Furthermore, PCBs are highly resistant to natural degradation (particularly the more chlorinated PCBs) and will persist for many decades, which could outlast the usefulness of the institutional controls (which individuals already appear to ignore) or the ability of AK Steel to enforce the institutional controls now in place. Also, various deficiencies and irregularities were noted in the field notes of the HUS, which make the results of the HUS—such as limitations regarding the ability to identify repeat recreators at Dick's Creek—suspect.

5.0 Toxicity Assessment

PCBs are well absorbed from the gastrointestinal tract, skin, and lungs. PCBs initially concentrate in the liver, blood, and muscle, but are soon sequestered into fat tissue, where they have a long half life, typically on the order of decades. PCBs are metabolized to biphenyls, biphenyldiols, and dihydrodihydroxybiphenyls, and are ultimately excreted in urine and feces. Although there are species variations, the more highly chlorinated compounds are excreted more in the feces and are less readily metabolized than are less-chlorinated isomers.

Animal studies reveal a considerable variation in equipotent doses between species of both animals and PCBs. In comparable studies, however, the more chlorinated mixtures are more toxic than the less chlorinated ones. This trend predominantly holds between LD50 and carcinogenicity studies.

In humans, the primary acute toxic effect of PCBs is chloracne. Chronic ingestion of PCBs causes Yusho Disease, named after the town of Yusho, Japan, where an epidemic occurred when residents ate PCB-contaminated food for several months. Chloracne develops after a latent period, along with pigmentation of skin areas, visual disturbances, gastrointestinal distress, jaundice, and lethargy. Infants from exposed mothers had low birth weight and pigment blotches. Some of these effects, however, have been ascribed to the chemically related polychlorinated dibenzofurans (PCDFs), which are byproducts found in most complex mixtures of PCBs. Industrial exposure, which is generally limited to dermal contact, produces chloracne and, in severe cases, hepatotoxicity. PCBs produce reproductive toxicity based on results of the few animal studies; the Yusho incident; and, more recently, a similar incident in Taiwan.

The systemic (noncancer) effects are represented by the reference dose (RfD). The RfD for Aroclor 1012 is 7E-5 mg/kg-day based on the toxic effect of reduced birth weights. For Aroclor 1254, the RfD is slightly lower at 2E-5 mg/kg-day and is based on the toxic effects of ocular exudate (eye secretions), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails, and decreased antibody (IgG and IgM) response to sheep erythrocytes.

PCBs are class B2, or probable human carcinogens, based on the induction of liver tumors in experimental animals (USEPA 1995).

Unlike conventional risk assessments, where specific toxicity values are developed for individual chemicals, Aroclors are complex mixtures that, once released into the environment, partition into different environmental media according to the physical-chemical properties of each PCB congener. That is, partitioning refers to processes by which different congeners fractionate or separate into water, sediment, and fish. As a general concept, the higher and more toxic chlorinated PCBs become concentrated into media with high organic content (such as sediments and fish) and, conversely, congeners with low chlorine content tend to be more volatile and also more soluble in water. The USEPA PCB risk assessment methodology is based on this partitioning effect that distinguishes PCB mixtures by using information on partitioning of congeners in fate and transport environmental processes. Partitioning has profound effects that can decrease or increase toxicity in an individual medium, so toxicity of an environmental mixture is only partly determined by the original Aroclor commercial mixture. A PCB HHRA, therefore, requires a tiered approach where the toxicity value used is dependent on the environmental medium and exposure pathway, rather than the Aroclor that is detected in the medium (as was the case with both TT and AGM HHRAs). As indicated in Table 7, the highest observed potency from these ranges is appropriate for food chain exposure, sediment or soil ingestion, and dust or aerosol inhalation—pathways where environmental processes tend to increase risk. Lower potencies are appropriate for ingestion of water-soluble congeners or inhalation of evaporated congeners—pathways where environmental processes tend to decrease risk. To the extent that drinking water or ambient air contains contaminated sediment or dust, the higher potency values would be appropriate, as congeners adsorbed to sediment or dust tend to be of high chlorine content and persistence, especially for sediment or dust with high organic content. This updated HHRA follows this PCB risk assessment methodology (USEPA 1996) which is presented in Table 7.

TABLE 7

TIERS OF HUMAN CANCER SLOPE FACTORS FOR ENVIRONMENTAL PCB MIXTURES BASED ON EXPOSURE ROUTES

HIGH RISK AND PERSISTENCE				
ED10	**LED10**	**Central Slope Factor**	**Upper-Bound Slope Factor**	**Exposure Pathways**
0.086	0.067	1	2	Food chain exposure Sediment or soil ingestion Dust or aerosol inhalation Dermal exposure, if an absorption factor has been applied to reduce the external dose Presence of dioxin-like, tumor-promoting, or persistent congeners in other media Early-life exposure (all pathways and mixtures)
LOW RISK AND PERSISTENCE				
ED10	**LED10**	**Central Slope Factor**	**Upper-Bound Slope Factor**	**Exposure Pathways**
0.38	0.27	0.3	0.4	Ingestion of water-soluble congeners Inhalation of evaporated congeners Dermal exposure, if no absorption factor has been applied to reduce the external dose
LOWEST RISK AND PERSISTENCE				
ED10	**LED10**	**Central Slope Factor**	**Upper-Bound Slope Factor**	**Exposure Pathways**
2.4	1.4	0.04	0.07	Congener or isomer analyses verify that congeners with more than 4 chlorines constitute less than 0.5% of total PCBs

Notes: ED10: Estimated dose associated with 10% increased incidence, in mg/kg-d
LED10: 95% lower bound on ED10, in mg/kg-d
Central Slope: per mg/kg-d, computed as 0.10/ED10 and rounded to one significant digit
Upper-Bound Slope: per mg/kg-d, computed as 0.10/LED10 and rounded to one significant digit

The last departure from the conventional risk assessment approach for single chemicals is the use of central-estimate slope factors in PCB risk assessments. These are derived by linear extrapolation from ED10s, which can be described by a similar range with three reference points. Central-estimate slope factors are used to estimate a typical individual's risk, while upper bound slope factors assure that this risk is not likely to be underestimated if the underlying model is correct. In this updated HHRA, both central tendency exposure (CTE) and RME risks were calculated with both upper-bound and central tendency slope factors.

It should be stressed that commercial Aroclors tested in laboratory animals for the inherent toxicity of each Aroclor mixture were not subject to prior selective retention of persistent congeners through the food chain. This is important because bioaccumulated PCBs, such as those ingested through the fish ingestion pathway, appear to be more toxic than commercial PCBs and are more persistent in the body. Although no methodology yet exists for determining the empirical increase in toxicity on a site-specific basis through exposure through the food chain, it is reasonable to conclude risks from fish ingestion are higher than those estimated in this updated HHRA. In addition, because PCBs persist for a long period in the body, they provide a continuing source of internal exposure after external exposure stops (which is why it is important to include pre-1998 data). There may be greater-than-proportional effects from less-than-lifetime exposure, especially for persistent mixtures and for early-life exposures.

No effort was made in this updated HHRA to specifically evaluate sensitive populations for whom the risk estimates may not apply. These individuals would include nursing infants, particularly in those families who consume fish from Dick's Creek, as well as those with decreased liver function (USEPA 1996). In early-life exposure, infants can be highly exposed to PCBs during pregnancy and lactation (Dewailly *et al.* 1991, 1994). The accumulation of PCBs in human adipose tissue creates a store for subsequent release of PCBs into the bloodstream and then into the fetal circulation. During the postpartum period, PCBs are mobilized from adipose stores, transferred into human milk, and delivered to the neonate via nursing. This source of exposure may account for a substantial fraction of chlorinated PCBs. USEPA suggests that an assessment be made of the extent of exposure through the human milk pathway; if direct measurement of concentrations in milk are not available, estimates can be derived from modeling maternal-to-infant exposures (Smith 1987). However, the constraints of this study did not allow such an analysis.

As mentioned previously, one of the most significant omissions in the comprehensive database is the absence of dioxin-like PCB congeners (in addition to concluding no PCBs are present in samples based on non-detects for Aroclors). A small group of 12 PCB congeners produce dioxin-like effects. These dioxin-like effects are toxicologically identical to dioxin (2,3,7,8-tetrachlorodibenzo-*p*-dioxin [TCDD]) itself, which USEPA considers to be highly toxic and carcinogenic:

> *"When assessing PCB mixtures, it is important to recognize that both dioxin-like and nondioxin-like modes of action contribute to overall PCB toxicity (Safe, 1994; McFarland and Clarke, 1989; Birnbaum and DeVito, in press). Because relatively*

> *few PCB congeners are dioxin-like, dioxin equivalence explains only part of a PCB mixture's toxicity."*

Like USEPA, the National Academy of Sciences, NRC committee (NRC 2001) strongly emphasizes the need for analyzing for PCB congeners to calculate risks associated with dioxin-like PCBs, stating:

> *"The non- and mono-ortho-substituted PCBs are of particular concern, because these congeners can assume a planar or nearly planar conformation similar to that of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (Safe 1990; Giesy et al. 1994a; Metcalfe and Haffner 1995) and have toxic effects similar to TCDD."*

At many hazardous waste sites, the human health risks associated with dioxin-like PCB congeners is significantly greater and of much greater health concern than nondioxin-like PCBs. USEPA provides a case example of this in its PCB risk assessment guidance (USEPA 1996) and has also developed a protocol for quantifying the risks based on Toxicity Equivalence Factors (TEQs):

> *"When assessing mixtures of dioxin and related compounds, it is important to consider the contribution of dioxin-like PCBs to total dioxin equivalents (U.S. EPA, 1994b). TEQs for dioxin-like PCBs (Ahlborg et al., 1994) can be added to those for other dioxin-like compounds. In some situations, PCBs can contribute more dioxin-like toxicity than chlorinated dibenzo-p-dioxins and dibenzofurans (Schecter et al., 1994; Dewailly et al. 1991, 1994). The congener 2,4,5,3',4'-pentachlorobiphenyl, shown to have tumor-promoting activity, is a major contributor to total dioxin equivalents in the United States (Patterson et. al., 1994) and maritime Quebec (Dewailly et al. 1994)."*

Although dioxin-like PCB risks were estimated for fish ingestion in the TT HHRA, they could not be included in this update because the quality of the data has not been confirmed. However, it is likely the risks from this small group of congeners would be high because they are found in relatively high concentrations in the Aroclor mixtures that were detected throughout sediment samples collected in Dick's Creek and its tributaries.

6.0 Risk Characterization

The final step in the updated HHRA is characterizing potential exposure and quantifying potential health hazards and carcinogenic risks associated with human contact with AK Steel's uncontrolled releases of PCBs into Dick's Creek and its tributaries. This step involves integrating the results of the data assessment (exposure point concentration), exposure assessment, and toxicity assessment presented in the preceding sections.

In the first stage of risk characterization, PCBs in sediments, surface water, and fish are organized into groups of carcinogenic and noncarcinogenic exposures for individual exposure pathways. This step is carried out for the child (0-6 years), adolescent (7-12 years), and adult. Numerical estimates of carcinogenic and noncarcinogenic risk are calculated separately. The overall risks for current and hypothetical future receptors are derived by summing hazard quotients and carcinogenic risks for each exposed population across individual exposure routes.

Risks associated with exposure to potential human carcinogens are estimated as the incremental probability of an individual developing cancer over a lifetime as a direct result of exposure to the chemical (USEPA 1989). The estimated risk is expressed as a unitless probability. For instance, a probability of 1E-06 indicates that one individual in one million may develop cancer during a 70-year lifetime as a result of the defined exposure conditions when exposed to the chemical over a thirty-year exposure period.

Carcinogenic risks are additive. Thus, total excess lifetime carcinogenic risk associated with an exposure pathway were calculated by adding the individual risk from each Aroclor mixture, beginning with the point in time when PCB data became available (pre-1998 data have been weighted to establish a temporal trend). Likewise, the overall risks to an exposed population were derived by aggregating carcinogenic risks from all pertinent exposure pathways for each age of the receptor group. Based on current census data, the mean and upper 95th percentiles for a resident staying in the same home are 9 years and 30 years, respectively. Likewise, it is assumed that a receptor who resides near Dick's Creek will frequently be exposed (due to its close proximity-within walking distance) to Dick's Creek and its tributaries over the period the receptor lives in the same home. For this reason, it is assumed that a lifetime exposure for a resident could begin with childhood exposures, with the person continually exposed as they mature into an adult. Clearly, the exposure would end when the person no longer resides in the region and moves away, which is expected to occur after 9 and 30 years. Accordingly, a total lifetime exposure to Dick's Creek is assumed to last either 9 years or 30 years, representing the CTE or RME. It is also assumed that recreational fisherman do not catch fish and selfishly prepare and consume them alone, but take their catch and share their daily catch of fish with others, which may include children their family.

Noncarcinogenic adverse effects produced by PCBs were estimated by dividing the average daily dose by the USEPA-derived RfDs. RfDs are fundamentally different from cancer slope factors (CSFs) in several respects. Carcinogenic chemicals are presumed to produce nonthreshold carcinogenic effects at any dose level, whereas noncarcinogenic effects are assumed to have a threshold. Noncarcinogenic effects are only manifest when a threshold is exceeded. At subthreshold doses, no evidence of damage is observed; accordingly, toxicologists have termed this concentration as the no observed adverse effect level (NOAEL). The NOAEL is typically based on the most sensitive animal species, and is used to develop the RfD, which is the NOAEL multiplied by uncertainty and modifying factors. Modifying factors are necessary to account for both intra- and interspecies variation, metabolic and pharmacokinetic mechanisms between animal and humans, and different experimental paradigms.

Tables 8 through 12 present the CTE noncarcinogenic health hazards (both Hazard Quotients and Hazard Index) and carcinogenic risk using both the RME and CTE slope factors discussed above. Tables 13 through 17 present the RME for the same results. It should be noted that the carcinogenic risks were calculated based on total PCBs detected in each sample (i.e., Aroclors were summed for the sample), whereas health hazards were calculated for individual Aroclors (because they have different RfD values) and the hazard quotients summed.

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TABLE 8

SUMMARY CENTRAL TENDENCY EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 2 (DC 2)

Receptor	Exposure Pathway	Hazard Quotient	Carcinogenic Risk	
			RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	1.32e-01	5.06e-07	2.53e-07
	Sediment dermal contact	1.16e-01	4.47e-07	2.23e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	2.84e+02	1.14e-03	5.68e-04
	Cumulative totals	2.84e+02	1.14e-03	5.68e-04
Child Recreator (0 to 6)	Sediment ingestion	1.23e+00	1.35e-06	6.77e-07
	Sediment dermal contact	2.57e-01	2.83e-07	1.41e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	4.80e+02	5.46e-04	2.73e-04
	Cumulative totals	4.81e+02	5.48e-04	2.74e-04
Adolescent Recreator (7 to 12)	Sediment ingestion	5.58e-01	6.13e-07	3.06e-07
	Sediment dermal contact	1.88e-01	2.07e-07	1.03e-07
	Surface water ingestion	0.00e+00	0.00e+00	1.69e-03
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	7.46e-01	8.20e-07	1.69e-03
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			1.69e-03	8.42e-04
Hazard Index		7.66e+02		

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TABLE 9

SUMMARY CENTRAL TENDENCY EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 3 (DC 3)

Receptor	Exposure Pathway	Hazard Quotient	Carcinogenic Risk	
			RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	1.33e-02	4.14e-08	2.07e-08
	Sediment dermal contact	1.18e-02	3.65e-08	1.83e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	2.51e-02	7.79e-08	3.90e-08
Child Recreator (0 to 6)	Sediment ingestion	1.24e-01	1.11e-07	5.54e-08
	Sediment dermal contact	2.60e-02	2.31e-08	1.16e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	1.50e-01	1.34e-07	6.70e-08
Adolescent Recreator (7 to 12)	Sediment ingestion	5.66e-02	5.01e-08	2.51e-08
	Sediment dermal contact	1.91e-02	1.69e-08	8.46e-09
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	7.57e-02	6.70e-08	3.36e-08
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			2.12e-07	1.06e-07
Hazard Index		1.75e-01		

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TABLE 10

SUMMARY CENTRAL TENDENCY EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 4 (DC 4)

Receptor	Exposure Pathway	Hazard Quotient	Carcinogenic Risk	
			RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	1.34e-02	4.20e-08	2.10e-08
	Sediment dermal contact	1.18e-02	3.70e-08	1.85e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	7.70e+01	8.57e-05	4.29e-05
	Cumulative totals	7.70e+01	8.58e-05	4.29e-05
Child Recreator (0 to 6)	Sediment ingestion	1.25e-01	1.12e-07	5.61e-08
	Sediment dermal contact	2.61e-02	2.34e-08	1.17e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	1.30e+02	4.12e-05	2.06e-05
	Cumulative totals	1.30e+02	4.13e-05	2.07e-05
Adolescent Recreator (7 to 12)	Sediment ingestion	5.67e-02	5.08e-08	2.54e-08
	Sediment dermal contact	1.91e-02	1.71e-08	8.57e-09
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	7.58e-02	6.79e-08	3.40e-08
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			1.27e-04	6.36e-05
Hazard Index		2.07e+02		

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TABLE 11

SUMMARY CENTRAL TENDENCY EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 5 (DC 5)

Receptor	Exposure Pathway	Hazard Quotient	Carcinogenic Risk	
			RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	2.21e-03	8.65e-09	4.32e-09
	Sediment dermal contact	1.95e-03	7.63e-09	3.82e-09
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	4.16e-03	1.63e-08	8.14e-09
Child Recreator (0 to 6)	Sediment ingestion	2.06e-02	2.31e-08	1.16e-08
	Sediment dermal contact	4.31e-03	4.83e-09	2.42e-09
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	2.49e-02	2.79e-08	1.40e-08
Adolescent Recreator (7 to 12)	Sediment ingestion	9.36e-03	1.05e-08	5.24e-09
	Sediment dermal contact	3.16e-03	3.53e-09	1.77e-09
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	1.25e-02	1.40e-08	7.01e-09
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			4.42e-08	2.22e-08
Hazard Index		2.91e-02		

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TABLE 12

SUMMARY CENTRAL TENDENCY EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

LANDFILL TRIBUTARY

Receptor	Exposure Pathway	Hazard Quotient	Carcinogenic Risk	
			RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	1.39e-01	3.97e-07	1.99e-07
	Sediment dermal contact	1.23e-01	3.51e-07	1.75e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	2.62e-01	7.48e-07	3.74e-07
Child Recreator (0 to 6)	Sediment ingestion	1.30e+00	1.06e-06	5.31e-07
	Sediment dermal contact	2.71e-01	2.22e-07	1.11e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	1.57e+00	1.28e-06	6.42e-07
Adolescent Recreator (7 to 12)	Sediment ingestion	5.89e-01	4.81e-07	2.41e-07
	Sediment dermal contact	1.99e-01	1.62e-07	8.12e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	7.88e-01	6.43e-07	3.22e-07
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			2.03e-06	1.02e-06
Hazard Index		1.83e+00		

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TABLE 13

SUMMARY REASONABLE MAXIMUM EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 2 (DC 2)

Receptor	Exposure Pathway	Hazard Quotient	RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	4.98e-01	6.57e-06	3.28e-06
	Sediment dermal contact	3.86e-01	5.08e-06	2.54e-06
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	1.02e+03	1.40e-02	1.40e-02
	Cumulative totals	1.02e+03	1.40e-02	1.40e-02
Child Recreator (0 to 6)	Sediment ingestion	4.66e+00	1.53e-05	7.65e-06
	Sediment dermal contact	8.72e-01	2.88e-06	1.44e-06
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	1.70e+03	5.82e-03	2.91e-03
	Cumulative totals	1.71e+03	5.84e-03	2.92e-03
Adolescent Recreator (7 to 12)	Sediment ingestion	2.11e+00	6.96e-06	3.48e-06
	Sediment dermal contact	6.15e-01	2.02e-06	1.01e-06
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	2.73e+00	8.98e-06	4.49e-06
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			1.99e-02	1.69e-02
Hazard Index		2.73e+03		

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TABLE 14

SUMMARY REASONABLE MAXIMUM EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 3 (DC 3)

Receptor	Exposure Pathway	Hazard Quotient	RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	5.05e-02	5.37e-07	2.69e-07
	Sediment dermal contact	3.92e-02	4.16e-07	2.08e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	8.97e-02	9.53e-07	4.77e-07
Child Recreator (0 to 6)	Sediment ingestion	4.73e-01	1.25e-06	6.26e-07
	Sediment dermal contact	8.85e-02	2.36e-07	1.18e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	5.62e-01	1.49e-06	7.44e-07
Adolescent Recreator (7 to 12)	Sediment ingestion	2.14e-01	5.70e-07	2.85e-07
	Sediment dermal contact	6.24e-02	1.66e-07	8.28e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	2.76e-01	7.36e-07	3.68e-07
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			2.44e-06	1.22e-06
Hazard Index		6.51e-01		

TABLE 15

SUMMARY REASONABLE MAXIMUM EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 4 (DC 4)

Receptor	Exposure Pathway	Hazard Quotient	RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	5.06e-02	5.44e-07	2.72e-07
	Sediment dermal contact	3.93e-02	4.21e-07	2.11e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	1.30e-02	1.06e-03	5.29e-04
	Cumulative totals	1.03e-01	1.06e-03	5.29e-04
Child Recreator (0 to 6)	Sediment ingestion	4.74e-01	1.27e-06	6.34e-07
	Sediment dermal contact	8.88e-02	2.39e-07	1.19e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	7.81e-03	4.39e-04	2.20e-04
	Cumulative totals	5.71e-01	4.41e-04	2.21e-04
Adolescent Recreator (7 to 12)	Sediment ingestion	2.15e-01	5.77e-07	2.89e-07
	Sediment dermal contact	6.26e-02	1.68e-07	8.39e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	2.78e-01	7.45e-07	3.73e-07
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			**1.50e-03**	**7.50e-04**
Hazard Index		**6.74e-01**		

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TABLE 16

SUMMARY REASONABLE MAXIMUM EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

DICK'S CREEK SEGMENT 5 (DC 5)

Receptor	Exposure Pathway	Hazard Quotient	RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	8.35e-03	1.12e-07	5.61e-08
	Sediment dermal contact	6.48e-03	8.69e-08	4.34e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	1.48e-02	1.99e-07	9.95e-08
Child Recreator (0 to 6)	Sediment ingestion	7.82e-02	2.61e-07	1.31e-07
	Sediment dermal contact	1.46e-02	4.93e-08	2.46e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	9.28e-02	3.10e-07	1.56e-07
Adolescent Recreator (7 to 12)	Sediment ingestion	3.55e-02	1.19e-07	5.95e-08
	Sediment dermal contact	1.03e-02	3.46e-08	1.73e-08
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	4.58e-02	1.54e-07	7.68e-08
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			5.09e-07	2.55e-07
Hazard Index		1.08e-01		

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TABLE 17

SUMMARY REASONABLE MAXIMUM EXPOSURE: NONCARCINOGENIC HAZARDS AND CARCINOGENIC RISKS USING RME AND CTE SLOPE FACTORS

LANDFILL TRIBUTARY

Receptor	Exposure Pathway	Hazard Quotient	RME Slope Factor	CTE Slope Factor
Adult Recreator	Sediment ingestion	5.25e-01	5.16e-06	2.58e-06
	Sediment dermal contact	4.08e-01	3.99e-06	2.00e-06
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	9.33e-01	9.15e-06	4.58e-06
Child Recreator (0 to 6)	Sediment ingestion	4.92e+00	1.20e-05	6.00e-06
	Sediment dermal contact	9.21e-01	2.26e-06	1.13e-06
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	0.00e+00	0.00e+00	0.00e+00
	Cumulative totals	5.84e+00	1.43e-05	7.13e-06
Adolescent Recreator (7 to 12)	Sediment ingestion	2.23e+00	5.47e-06	2.73e-06
	Sediment dermal contact	6.49e-01	1.59e-06	7.95e-07
	Surface water ingestion	0.00e+00	0.00e+00	0.00e+00
	Surface water dermal contact	0.00e+00	0.00e+00	0.00e+00
	Fish Ingestion	NA	NA	NA
	Cumulative totals	2.88e+00	7.06e-06	3.53e-06
Lifetime Exposure (Child and Adult Exposure)				
Carcinogenic Risk			2.34e-05	1.17e-05
Hazard Index		6.77e+00		

7.0 Uncertainty Associated with Risk Estimates

The discussion of uncertainty is an important component of the risk assessment because there are varying degrees of uncertainty at each stage of the HHRA analysis. Uncertainty can either be quantified with probabilistic methods (i.e., Monte Carlo simulations) or qualitatively by identifying sources of uncertainty that can result in over- and under-prediction of adverse health effects and carcinogenic risk. Probabilistic methods are outside the scope of this update. Therefore, the following are the major potential sources that could result in an underestimate of risk in this update:

- The lack of congener-specific data that could render non-detect values meaningless (PCBs could be present in the sample);
- The lack of congener-specific data that prevented dioxin-like risks from being calculated in sediments, surface water, and fish, which could lead to significant underestimation;
- Unknown sources of PCBs yet to be identified in Dick's Creek and its tributaries;
- Not modeling potential risks to nursing infants who may be indirectly exposed through breast-feeding females; and
- Not evaluating the increase in inherent toxicity of bioaccumulated PCBs.

In contrast, the following are the major potential sources that could result in an overestimation of risk in this update:

- Overestimating the amount of fish consumed by individuals living in the area; and
- Assuming all PCBs detected in fish (total PCBs were used in the update) originated from uncontrolled releases associated with the AK facility rather than other ambient anthropogenic background sources.

Other sources of uncertainty that would have uncertain consequences on the risk estimates include the following:

- Unknown differences between humans and laboratory animals with regard to the absorption, distribution, metabolism, excretion and overall toxicity of PCB congeners;
- Validity and quality of scientific studies that form the basis of EPA-derived toxicity values;
- Derivation of absorbed dose from oral toxicity values to estimate dermal absorption; and

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- Statistical models used to extrapolate from high to low doses in animal studies.

8.0 References

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APPENDIX A

Exposure Parameter Values

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
CHILD RECREATOR (AGED 0 TO 6)

Exposure Medium: Sediment
Receptor: Child Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (mg/day), note c	200	100	Exposure=C*IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)	Exposure=C*IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	CF (kg/mg)	1.00E-06	1.00E-06		
	FI (unitless), e	1	0.5		
	BW (kg), p	15	15		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		
Dermal	SA (cm2), h	894	498	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)
	AF (mg/cm2-day), i	0.3	0.3		
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	CF (kg/mg)	1.00E-06	1.00E-06		
	BW (kg), p	15	15		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
CHILD RECREATOR (AGED 0 TO 6)

Exposure Medium: Surface Water Receptor: Child Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (L/day), note b	0.1	0.05	Exposure =IR*Ao*EF*ED*(1/BW)*(1/AT)	Exposure =IR*Ao*EF*ED*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	BW (kg), p	15	15		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		
Dermal	SA (cm2), g	5885	5327	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	ET (hrs/day), k	2	1		
	CF (L/cm3)	1.00E-03	1.00E-03		
	BW (kg), p	15	15		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
ADOLESCENT RECREATOR (AGED 7 TO 12)

Exposure Medium: Sediment
Receptor: Adolescent Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (mg/day), note c	200	100	Exposure =IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)	Exposure =IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	CF (kg/mg)	1.00E-06	1.00E-06		
	FI (unitless), e	1	0.5		
	BW (kg), p	33	33		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		
Dermal	SA (cm2), h	1386	803	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)
	AF (mg/cm2-day), I	0.3	0.3		
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	CF (kg/mg)	1.00E-06	1.00E-06		
	BW (kg), p	33	33		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
ADOLESCENT RECREATOR (AGED 7 TO 12)

Exposure Medium: Surface Water
Receptor: Adolescent Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (L/day), b	0.1	0.05	Exposure =IR*Ao*EF*ED*(1/BW)*(1/AT)	Exposure =IR*Ao*EF*ED*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	BW (kg), p	33	33		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		
Dermal	SA (cm2), g	9102	8303	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)
	EF (days/year), m	89	47		
	ED (years), o	6	2		
	ET (hrs/day), k	2	1		
	CF (L/cm3)	1.00E-03	1.00E-03		
	BW (kg), p	33	33		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
ADULT RECREATOR

Exposure Medium: Sediment
Receptor: Adult Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (mg/day), c	100	50	Exposure =IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)	Exposure =IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	89	47		
	ED (years), o	24	7		
	CF (kg/mg)	1.00E-06	1.00E-06		
	FI (unitless), e	1	0.5		
	BW (kg), p	70	70		
	ATc (days), q	25550	25550		
	ATnc (days), q	8760	2555		
Dermal	SA (cm2), h	1841	1050	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)
	AF (mg/cm2-day),	0.3	0.3		
	EF (days/year), m	89	47		
	ED (years), o	24	7		
	CF (kg/mg)	1.00E-06	1.00E-06		
	BW (kg), p	70	70		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	8760	2555		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
ADULT RECREATOR

Exposure Medium: Surface Water
Receptor: Adult Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (L/day), note b	0.037	0.01	Exposure =IR*Ao*EF*ED*(1/BW)*(1/AT)	Exposure =IR*Ao*EF*ED*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	89	47		
	ED (years), o	24	7		
	BW (kg), p	70	70		
	ATc (days), q	25550	25550		
	ATnc (days), q	8760	2555		
Dermal	SA (cm2), g	8595	6870	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)
	EF (days/year), m	89	47		
	ED (years), o	24	7		
	ET (hrs/day), k	2	1		
	CF (L/cm3)	1.00E-03	1.00E-03		
	BW (kg), p	70	70		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	8760	2555		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
CHILD FISH INGESTION (AGED 0 TO 6)

Exposure Medium: Fish Receptor: Child

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	CR (g/day), d	19.2	5.4	Exposure =CR*EF*ED*CF*FI*(1/BW)*(1/ATc)	Exposure =CR*EF*ED*CF*FI*(1/BW)*(1/ATc)
	EF (days/year), m	365	365		
	ED (years), o	6	2		
	CF (kg/g)	1.00E-03	1.00E-03		
	FI (unitless), e	1	0.05		
	BW (kg), p	15	15		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
ADULT FISH INGESTION

Exposure Medium: Fish
Receptor: Adult

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	CR (g/day), d	54	15	Exposure =CR*EF*ED*CF*FI*(1/BW)*(1/ATc)	Exposure =CR*EF*ED*CF*FI*(1/BW)*(1/ATc)
	EF (days/year), m	365	365		
	ED (years), o	24	7		
	CF (kg/g)	1.00E-03	1.00E-03		
	FI (unitless), e	1	0.05		
	BW (kg), p	70	70		
	ATc (days), q	25550	25550		
	ATnc (days), q	8760	2555		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
ADOLESCENT RECREATOR (AGED 7 TO 12)
LANDFILL TRIBUTARY

Exposure Medium: Sediment
Receptor: Adolescent Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (mg/day), note c	200	100	Exposure =IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)	Exposure =IR*Ao*EF*ED*CF*FI*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	21	15		
	ED (years), o	6	2		
	CF (kg/mg)	1.00E-06	1.00E-06		
	FI (unitless), e	1	0.5		
	BW (kg), p	33	33		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		
Dermal	SA (cm2), h	1386	803	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*AF*EF*ED*CF*ABS*(1/BW)*(1/ATc)
	AF (mg/cm2-day), I	0.3	0.3		
	EF (days/year), m	21	10		
	ED (years), o	6	2		
	CF (kg/mg)	1.00E-06	1.00E-06		
	BW (kg), p	33	33		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		

ɔte: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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EXPOSURE PARAMETERS USED TO ESTIMATE PCB INTAKE (CHEMICAL DOSE)
ADOLESCENT RECREATOR (AGED 7 TO 12)
LANDFILL TRIBUTARY

Exposure Medium: Surface Water
Receptor: Adolescent Recreator

Exposure Route	Parameter Code	RME	CTE	Exposure Equation, RME	Exposure Equation, CTE
Ingestion	IR (L/day), b	0.095	0.01	Exposure =IR*Ao*EF*ED*(1/BW)*(1/AT)	Exposure =IR*Ao*EF*ED*(1/BW)*(1/ATc)
	Ao (unitless)	1	1		
	EF (days/year), m	21	10		
	ED (years), o	6	2		
	BW (kg), p	33	33		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		
Dermal	SA (cm2), g	2079	803	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)	Exposure =SA*EF*ED*ET*CF*ABS*(1/BW)*(1/ATc)
	EF (days/year), m	21	10		
	ED (years), o	6	2		
	ET (hrs/day), k	2	1		
	CF (L/cm3)	1.00E-03	1.00E-03		
	BW (kg), p	33	33		
	ABS, j	0.14	0.14		
	ATc (days), q	25550	25550		
	ATnc (days), q	2190	730		

Note: Chemical Intake (Average Daily Dose or Lifetime Average Daily Dose) = Exposure Point Concentration * Exposure

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Notes:

IR:	Ingestion rate
Ao:	Oral absorption
EF:	Exposure frequency
ED:	Exposure duration
CF:	Conversion factor
FI:	Fraction ingested from source
BW:	Body weight
ATc:	Averaging time (cancer)
ATnc:	Averaging time (noncancer)
SA:	Surface area
AF:	Adherence factor
ET:	Exposure time
ABS:	Absorption factor (chemical-specific)

cm/hr:	Centimeter per hour
cm^2:	Square centimeter
cm^2/day:	Square centimeter per day
CT:	Central tendency
EPC:	Exposure point concentration
FI:	Fraction ingested
g/day:	Gram per day
hrs/day:	Hours per day
IR:	Ingestion rate
kg:	Kilogram
L/cm^3:	Liter per cubic centimeter
L/day:	Liter per day
mg/cm^2:	Milligram per square centimeter
mg/cm^2 - event:	Milligram per square centimeter - event
mg/day:	Milligram per day
mg/kg:	Milligram per kilogram
mg/L:	Milligram per liter
RME:	Reasonable maximum exposure

NOTES ON EXPOSURE PARAMETER VALUES
HUMAN HEALTH RISK ASSESSMENT
AK STEEL FACILITY, MIDDLETOWN, OHIO

a Exposure parameter values are presented for the adolescent recreator (7 to 12) regarding potential exposures in Dataset Creek and in the Landfill Tributary. If a single value is presented in a cell, that value applies to potential exposures in both streams. If the exposure parameter values are different for exposure in the two streams, the first value is for the potential exposure in Dataset Creek, and the value in parentheses is for potential exposure in the Landfill Tributary.

b The RME and CT values for the adolescent recreator (7 to 12) in the Landfill Tributary assume that once per year, the receptor will ingest a mouthful of water. The water is likely to be unpalatable; thus, assuming more than one drink per year is unreasonable. It is assumed that 0.200 liter and 0.100 liter doses mouthfuls of water are ingested under the RME and CT scenarios, respectively. These quantities are divided by the exposure frequency to obtain the IR_{sw} values presented: (0.200 L)/(21 day) = 0.095 L/day; (0.100 L)/(10 day) = 0.010 L/day. (Also see note m.)

Child recreator (0 to 6) and adolescent recreator (7 to 12) values assume that 0.05 L/hour will be ingested under the RME and CT scenarios during swimming. Therefore: (0.05 L/hr)*(2 hr/day) = 0.100 L/day; (0.05 L/hr)*(1 hr/day) = 0.050 L/day. As discussed in footnote h, adult recreators are assumed to swim 37 percent (33/89 days) of the days they are potentially exposed under RME conditions and 21 percent (10/47 days) of the days they are potentially exposed under CT conditions. Therefore, because ingestion of surface water is assumed to occur only during swimming, the daily surface water ingestion rates are multiplied by factors of 0.37 and 0.21 under RME and CT conditions, respectively, to derive average daily IR_{sw} values of 0.037 and 0.01 L/day, respectively.

c Incidental ingestion rates are based on EPA (1991 and 1997). The values for the adult recreator are based on EPA (1991) and are consistent with EPA (1997). The values for the child recreator (0 to 6) and adolescent recreator (7 to 12) are based on several studies presented in EPA (1997).

d The adult recreator RME value was based on EPA (1991) for recreational fishers and compares favorably with the 95[th] percentile consumption rate of 58.11 grams per day presented in Table 10-63 of EPA (1997), and consequently provides for the possibility of non-Native American subsistence fishers. The adult recreator CT value was based on OEPA (1996). The child recreator (0 to 6) and adolescent recreator (7 to 12) RME and CT values are based on the adult recreator values multiplied by the ratio of consumption rates provided in Table 10-1 of EPA (1997; 95

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percentile for RMA and mean for CT conditions) for children aged 0 to 9 years and adults aged 20 years or older and for the average of children 0 to 9 years and children 10 to 19 years and adults aged 20 years or older, respectively.

e Estimates based on professional judgment. The total amount of fish ingested by the adult recreator under the RME scenario is calculated as 54 g/day x 365 days/year = 19,710 grams/year. An ounce is equivalent to 38.4 grams; therefore, 19,710 grams/year = 695.2 ounces/year = 13.4 ounces per week. Assuming that the recreator could catch 2 fish per week that each provided 2, 3.5-ounce filets, the assumption that FI = 1 is reasonable. Under the CT scenario, it is assumed that the recreators spend half their time at different fishing locations.

g All surface areas are from EPA 1997, Volume I, Tables 6-4 through 6-8. Surface area values for the adolescent recreator (7 to 12) potentially exposed in the Landfill Tributary assume exposure to feet and the lower leg and exposure to the feet only under RME and CT conditions, respectively. The surface area of the lower legs represents about 40 percent of the total surface area of the human leg (EPA 1997, Table 6-4). For adolescents 7 to 12 years of age, the legs represent about 28.8 percent of the total body surface area (11,000 cm^2; EPA 1997, Tables 6-2, 6-3, and 6-8). Therefore, the surface area for the lower leg was calculated as 11,000 cm^2 x 0.288 x 0.4 = 1,267 cm^2. For adolescents 7 to 12 years of age, the feet represent about 7.3 percent of the total body surface area, or 803 cm^2 (EPA 1997, Table 6-8). The total surface area assumed exposed under RME conditions (lower legs and feet) was calculated as 1,276 cm^2 + 803 cm^2 = 2,079 cm^2. Under CT conditions, only the feet (803 cm^2) were assumed to be exposed. All receptors potentially exposed in Dataset Creek are assumed to be exposed through a combination of swimming and wading activities. The total exposure frequency for child and adult recreators in Dataset Creek under RME and CT conditions is as follows: under RME conditions, exposure is assumed to occur 5 days/week for 13 weeks (June through August) and 6 days/month for 4 months (April, May, September, and October) for a total of 89 days. Under CT conditions, exposure is assumed to occur 3 days/week for 13 weeks and 2 days/month for 4 months for a total of 47 days.

Under RME conditions, child recreators are assumed to swim (whole body exposure) during the summer months and wade (exposure to legs and feet) during the remaining 4 months. Under CT conditions, child recreators are assumed to swim 75 percent of the summer days and wade the remainder of the available days. The total body surface areas for children (0 to 6) and adolescents (7 to 12) are 11,000 and 7,213 cm^2, respectively (based on an average of 50^{th} percentile values for boys and girls from Tables 6-6 and 6-7 in EPA 1992). Similarly, the surface area of legs and feet for children (0 to 6) and adolescents (7 to 12) were determined to be about 36 percent of the total body surface area (3,960 cm^2) and 31.7 percent of the total body surface area (2,287 cm^2), respectively. Therefore, the SAsw values for child recreators were determined as follows:

Child recreator (0 to 6):

$(7,213\ cm^2 \times 65/89) + (2,287\ cm^2 \times 24/89) = 5,885\ cm^2$ (RME conditions)

$(7,213\ cm^2 \times 29/47) + (2,287\ cm^2 \times 18/47) = 5,327\ cm^2$ (CT conditions)

Adolescent recreator (7 to 12):

$(11,000\ cm^2 \times 65/89) + (3,960\ cm^2 \times 24/89) = 9,102\ cm^2$ (RME conditions)

$(11,000\ cm^2 \times 29/47) + (3,960\ cm^2 \times 18/47) = 8,303\ cm^2$ (CT conditions)

By comparison, adult recreators were assumed to swim (whole body) 50 and 25 percent of the available summer days under RME and CT condition, respectively; wading was assumed on all other days. The total body surface areas for adults was identified as 18,000 cm^2 (based on an average of 50th percentile values for men and women (EPA 1992, Tables 6-2 and 6-2, and EPA 1999). Similarly, the surface area of lower legs and feet for adults was determined to be about 3,053 cm^2. Therefore, the SA_{sw} values for adult recreators were determined as follows:

Adult recreator:

$(18,000\ cm^2 \times 33/89) + (3,053\ cm^2 \times 56/89) = 8,595\ cm^2$ (RME conditions)

$(18,000\ cm^2 \times 12/47) + (3,053\ cm^2 \times 35/47) = 6,870\ cm^2$ (CT conditions)

h All surface areas are from EPA 1997, Volume I, Tables 6-4 through 6-8. RME values assume exposure to feet and hands, and CT values assume exposure to the feet only. For the child recreator (0 to 6), feet and hands represent about 12.4 percent of the total body surface area (7,213 cm^2) or about 894 cm2 under RME conditions. Similarly, the feet represent about 6.9 percent of the total body surface area or about 498 cm^2 under CT conditions. For the adolescent recreator (7 to 12), feet and hands represent about 12.6 percent of the total body surface area (11,000 cm^2) or about

1,386 cm^2 under RME conditions. Similarly, the feet represent about 7.3 percent of the total body surface area or about 803 cm^2 under CT conditions.

Finally, for the adult recreators, feet and hands (RME conditions) represent about 1,841 cm^2 and feet (CT conditions) represent about 1,050 cm^2 (Table 6-4).

i The soil adherence value recommended for children in EPA Region 9 Preliminary Remediation Goals (PRGs) 1999 is 0.2 mg/cm^2 (EPA 1999b). The increase from 0.2 to 0.3 mg/cm^2 is based on professional judgment and is intended to account for additional adherence due to the "sticky" nature of some sediments. The adherence values for "kids-in-mud" from EPA's "Exposure Factors Handbook" were judged to be overly conservative given the often high sand and low clay content of stream sediments in the study area and the potential for sediment to be washed from body parts due to contact with surface water (EPA 1997b). The adherence value for children was conservatively used to also represent adults.

j Chemical-specific absorption factor; 0.14 was used for dermal absorption of PCBs (Aroclors), in accordance with values obtained from USEPA Region 5 Dermal Workgroup Staff (Tetra Tech 1998c).

k Estimated based on professional judgment.

m Exposure frequency for the adolescent recreators (7 to 12) exposed in the Landfill Tributary under RME conditions was calculated based on the assumption of 1 day/week for June, July, and August and 2 days/month for April, May, September, and October. Under CT conditions, it was assumed that exposure took place 2 days/month for June, July, and August and 1 day/month for April, May, September, and October. See footnote g for the basis of receptor-specific EF values for exposure in Dick's Creek.

n Fish ingestion rates are daily rates averaged over an entire year (365 days).

o Exposure durations were obtained from EPA (1991).

p The body weight for the child recreator is the default child body weight (EPA 1989). The body weight for the adolescent recreator (7 to 12) was calculated as the average body weight for boys and girls at least 6 years old, but younger than 13 years (EPA 1997, Table 7-3). The body weight for adult recreators is the standard default adult body weight (EPA) 1989).

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q The averaging time for noncarcinogens reflects the exposure durations of 2, 6, 7, and 24 years: 2 years x 365 days/year = 730 days; 6 years x 365 days/year = 2190 days; 7 years x 365 days/year = 2555 days; 24 years x 365 days/year = 8760 days (EPA 1989). The averaging time for carcinogens reflects a 70-year lifetime: 70 years x 365 days/year = 25550 days.